Emerging Biologic Therapies in Autoimmune Diseases: Focus on Pemphigus Vulgaris, Generalized Myasthenia Gravis, and Psoriasis

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ABSTRACT

Biologic therapies have transformed the management of autoimmune diseases by targeting key immune pathways with precision, reducing dependence on broad immunosuppression. This review explores advances in monoclonal antibody–based therapies across three representative autoimmune conditions: pemphigus vulgaris (PV), generalized myasthenia gravis (gMG), and psoriasis. In PV, B-cell depletion with rituximab has become the preferred first-line therapy, achieving durable remission and steroid-sparing outcomes. In gMG, complement inhibitors (eculizumab, ravulizumab, zilucoplan) and neonatal Fc receptor (FcRn) antagonists (efgartigimod, rozanolixizumab) provide rapid, clinically meaningful improvements in refractory patients. In psoriasis, therapies targeting the IL-23/IL-17 axis (guselkumab, risankizumab, bimekizumab) have achieved unprecedented levels of skin clearance and durability. Safety, accessibility, and biomarker-driven personalization remain challenges, while future directions include antigen-specific cell therapies, bispecific antibodies, and oral biologic mimetics. Collectively, these advances highlight the transformative role of biologics in autoimmune disease and the trajectory toward precision immunotherapy.

Keywords: Biologics, Monoclonal antibodies, Pemphigus vulgaris, Myasthenia gravis, Psoriasis, Autoimmune diseases, FcRn inhibitors, IL-23 inhibitors

INTRODUCTION

Monoclonal antibodies and biologic therapies have revolutionized the management of autoimmune diseases by enabling targeted immunomodulation rather than broad immunosuppression. These agents exploit diverse immune pathways, including cytokine-directed strategies (e.g., TNF-α and interleukin blockade), B-cell–directed depletion, integrin inhibition, and co-stimulation modulation [1]. The development of monoclonal antibodies and fusion proteins has transformed immunotherapy by allowing precise targeting of immune cell receptors and cytokines [1]. Contemporary approaches can be categorized into those that globally suppress immune responses versus those that promote “operational tolerance” — selectively attenuating pathogenic autoimmune responses while sparing protective immunity. Among these, TNF-α inhibitors and CD20-directed therapies represent landmark examples [1]. Such advances have demonstrated remarkable progress across conditions including rheumatoid arthritis, psoriasis, inflammatory bowel disease, ankylosing spondylitis, and juvenile idiopathic arthritis [1].

Building on this broad impact, monoclonal antibodies have also transformed outcomes in rare but severe autoimmune diseases that were historically difficult to manage. In pemphigus vulgaris (PV), B-cell–directed therapy with rituximab has emerged as a first-line standard, demonstrating superior remission rates and steroid-sparing effects compared with conventional immunosuppressants [4]. In generalized myasthenia gravis (gMG), biologics targeting the complement pathway (eculizumab, ravulizumab, zilucoplan) or blocking FcRn-mediated IgG recycling (efgartigimod, rozanolixizumab) have reshaped treatment algorithms for refractory disease [8–13]. In psoriasis, biologics targeting the IL-17 and IL-23 axes (bimekizumab, guselkumab,

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risankizumab) have set new efficacy benchmarks, achieving durable skin clearance with favourable safety [14–20]. Collectively, these examples illustrate the paradigm shift from generalized immunosuppression to precision immunotherapy tailored to specific immune mechanisms.

Pemphigus Vulgaris (PV)

Pathogenesis and Rationale

Pemphigus vulgaris (PV) is a rare but potentially life-threatening autoimmune blistering disease in which IgG autoantibodies target desmoglein-1 and desmoglein-3, disrupting keratinocyte adhesion and leading to acantholysis and mucocutaneous blistering. Traditional therapy has relied on high-dose corticosteroids and broad immunosuppressants (azathioprine, cyclophosphamide, mycophenolate mofetil), which reduce disease activity but are associated with substantial morbidity, including opportunistic infections, osteoporosis, metabolic complications, and increased mortality. The recognition of B cells as central drivers of PV pathogenesis provided the rationale for B-cell–directed biologics as disease-modifying agents.

Rituximab as a Paradigm Shift

Rituximab, a chimeric anti-CD20 monoclonal antibody, represented the first major breakthrough. Early case series and observational studies suggested efficacy in inducing long-term remission. The pivotal PEMPHiX trial [4] provided high-level evidence by comparing rituximab with mycophenolate mofetil in patients with moderate-to-severe PV. Rituximab achieved sustained complete remission in 40% of patients at 52 weeks versus only 10% with MMF, while also enabling significant corticosteroid sparing. These findings firmly established rituximab as first-line therapy for PV.

Further support came from the Ritux 3 regimen [5], which demonstrated that early introduction of rituximab plus short-course corticosteroids resulted in long-lasting remission, with many patients remaining steroid-free for years. These results highlight how biologics have transformed PV management from chronic steroid dependence to durable, targeted disease control.

Safety Profile

Rituximab is generally well tolerated. The most common adverse events are infusion reactions and mild infections. Rare but serious risks include hepatitis B reactivation and progressive multifocal leukoencephalopathy (PML), underscoring the need for screening and pharmacovigilance. Long-term follow-up studies indicate that repeat courses remain safe and effective, with no major cumulative toxicity reported.

Emerging Therapies

The limitations of rituximab—delayed onset and incomplete remission in some patients—have prompted exploration of novel therapies. Rilzabrutinib, a Bruton's tyrosine kinase (BTK) inhibitor, demonstrated encouraging efficacy in phase 2 trials, but the PEGASUS phase 3 study failed to meet its primary endpoint, highlighting challenges in translating preclinical success to robust clinical outcomes. Other promising investigational strategies include desmoglein-specific CAR-T cell therapy (DSG3-CAART), which seeks to selectively eliminate autoreactive B cells, and next-generation anti-CD20 mAbs (ocrelizumab, ofatumumab) with potentially improved safety profiles.

Table 1: Key Biologic Therapies in Pemphigus Vulgaris (PV)

Agent	Target	Pivotal Trial(s)	Population / Endpoints	Key Efficacy Results	Safety Profile
Rituximab	CD20 B cells	PEMPHiX (NEJM 2021)	Moderate–severe PV, rituximab vs	Sustained complete remission at week 52: 40% vs 10% (MMF); reduced	Infusion reactions, viral reactivation (HBV), rare

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			MMF	cumulative steroids	PML
Rituximab (Ritux 3 regimen)	CD20 B cells	JAMA Derm 2024	Newly diagnosed PV	Long-term steroid-free remission (up to 5 years)	Similar to above; generally well tolerated with repeat dosing
Rilzabrutinib	BTK	PEGASUS Phase 3 (2022)	Moderate–severe PV	Did not meet primary endpoint; some secondary signals	Headache, GI symptoms; no major safety signals
Emerging: DSG3-CAART / CAR-T	Autoantigen-specific	RESET-PV (early phase)	Refractory PV	Preclinical promise; early clinical trials ongoing	Still under investigation

Rituximab has fundamentally changed the treatment paradigm in PV, shifting care toward targeted, B-cell–directed immunotherapy that offers durable remission with reduced steroid exposure. The future lies in refining therapies to further enhance specificity and safety.

Generalized Myasthenia Gravis (gMG)

Pathogenesis and Rationale

Generalized myasthenia gravis (gMG) is an autoimmune neuromuscular disorder primarily caused by autoantibodies against the acetylcholine receptor (AChR), though some patients harbor antibodies against muscle-specific kinase (MuSK) or other antigens. These antibodies impair neuromuscular transmission, producing fatigable muscle weakness. Standard therapies—acetylcholinesterase inhibitors, corticosteroids, and immunosuppressants—provide partial and often nonspecific control. Advances in immunology identified complement activation and pathogenic IgG persistence as central mechanisms, leading to the development of biologics targeting these pathways.

Complement Inhibitors

- Eculizumab [8]: This terminal complement (C5) inhibitor demonstrated significant improvements in MG-ADL and QMG scores in refractory AChR+ gMG patients. Benefits appeared within weeks and were sustained with long-term therapy. However, the need for IV infusions every 2 weeks and the risk of meningococcal infection limited practicality.

- Ravulizumab [10]: A long-acting derivative of eculizumab, ravulizumab offers comparable efficacy with infusions every 8 weeks, greatly improving convenience. Its efficacy was consistent across subgroups, making it an attractive alternative.

- Zilucoplan [13]: A novel subcutaneous peptide inhibitor of C5, zilucoplan provides at-home dosing with robust improvements in MG-ADL and QMG, further broadening the complement blockade armamentarium.

FcRn Inhibitors

The neonatal Fc receptor (FcRn) regulates IgG recycling, prolonging the half-life of pathogenic antibodies. Its inhibition accelerates IgG degradation, reducing autoantibody burden.

- Efgartigimod [11]: An engineered Fc fragment antagonist, efgartigimod produced rapid, clinically meaningful improvements in MG-ADL, with individualized treatment cycles tailored to patient response.

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- Rozanolixizumab [12]: Administered subcutaneously, rozanolixizumab demonstrated significant improvements in MG-ADL and QMG in both AChR+ and MuSK+ patients, highlighting broad applicability.

Safety Profile

Complement inhibitors carry an increased risk of meningococcal infections, requiring vaccination and prophylaxis, while FcRn inhibitors are generally well tolerated, with headache and gastrointestinal symptoms most frequently reported.

Emerging Therapies

Other FcRn inhibitors (e.g., nipocalimab) and additional complement modulators are in late-phase development. B-cell–targeted therapy with rituximab shows clear benefit in MuSK+ MG, though results are inconsistent in AChR+ disease.

Table 2: Key Biologic Therapies in Generalized myasthenia gravis (gMG)

Agent	Target	Pivotal Trial(s)	Population / Endpoints	Key Efficacy Results	Safety Profile
Eculizumab	C5 complement	REGAIN (NEJM 2017)	AChR+ refractory gMG	Significant improvement in MG-ADL/QMG; rapid onset (weeks)	Meningococcal infection risk; requires vaccination
Ravulizumab	C5 complement (long-acting)	CHAMPION-MG (2022)	AChR+ gMG	Non-inferior to eculizumab; q8-week dosing; durable benefit	Similar to eculizumab; fewer infusions
Zilucoplan	C5 peptide inhibitor (SC)	RAISE (Lancet Neurol 2023)	AChR+ gMG	Significant MG-ADL/QMG improvement; convenient SC dosing	Injection site reactions; generally well tolerated
Efgartigimod	FcRn antagonist	ADAPT (Lancet 2021)	AChR+ gMG	Rapid MG-ADL/QMG improvement; individualized cycles	Headache, mild infections; favorable safety
Rozanolixizumab	FcRn antagonist (SC)	MycarinG (Lancet Neurol 2023)	AChR+ and MuSK+ gMG	Significant MG-ADL/QMG improvement; weekly SC dosing	Nausea, headache; generally well tolerated
Rituximab (off-label)	CD20 B cells	Mixed trials / MuSK+ MG	MuSK+ MG, refractory cases	Effective in MuSK+ subtype; inconsistent AChR+ results	Infusion reactions, infections

The approval of complement and FcRn inhibitors has ushered in a new era of gMG management, enabling rapid, targeted, and durable symptom control while reducing dependence on broad immunosuppressants.

Psoriasis

Pathogenesis and Rationale

Psoriasis is a chronic, immune-mediated inflammatory disease of the skin, recognized globally as a serious non-communicable condition by the WHO due to its prevalence, morbidity, and psychosocial burden. Beyond

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skin manifestations, psoriasis is strongly associated with comorbidities such as psoriatic arthritis, cardiovascular disease, psychiatric disorders, and liver disease. Genetic predisposition is a major determinant, with over 60 susceptibility loci identified by genome-wide association studies and the HLA-Cw6 allele being particularly linked to early-onset disease.

Immunopathogenesis centers on dysregulated Th17 and Th22 responses, with cytokines such as IL-17, IL-22, IL-23, and TNF-α driving keratinocyte proliferation and chronic inflammation. Interactions between dendritic cells, antimicrobial peptides, and keratinocytes form a feedback loop that sustains disease activity. Clinically, psoriasis is heterogeneous, with multiple subtypes including plaque psoriasis, guttate, pustular, erythrodermic, scalp, nail, and palmoplantar psoriasis. This variability influences both disease burden and therapeutic selection.

Treatment Evolution

Traditional systemic therapies—methotrexate, cyclosporine, and acitretin—remain effective for moderate to severe psoriasis but are limited by toxicity and long-term tolerability. Phototherapy, including narrow-band UVB and PUVA, is a valuable adjunct for selected patients. The introduction of biologics targeting IL-17 (secukinumab, ixekizumab) and IL-12/23 (ustekinumab) represented a breakthrough, offering targeted immunomodulation with favourable efficacy and safety profiles. More recently, JAK inhibitors and personalized medicine approaches have emerged as promising strategies, emphasizing individualized care and integration of lifestyle modifications, patient education, and stress management into treatment planning.

Herbal and Natural Approaches

Alongside biologic therapies, recent research has highlighted the potential of natural products in psoriasis management. For instance, a 2024 study evaluated ointments containing hydro-alcoholic leaf extract of Cassia auriculata and demonstrated favourable physical properties, stability, and non-irritant profiles, supporting its traditional use in inflammatory skin disorders. The authors concluded that Cassia auriculata ointment may represent a promising adjunctive or alternative approach in psoriasis treatment. Such findings underscore the importance of integrating traditional medicinal plants into modern therapeutic frameworks, particularly in regions where access to biologics is limited. [30]

IL-17 and IL-23 Pathway Inhibitors

- Bimekizumab [17,18]: As a dual IL-17A/F inhibitor, bimekizumab demonstrated superior PASI 90/100 responses compared to adalimumab and secukinumab, setting new benchmarks for efficacy. Oral candidiasis was more frequent but typically mild and manageable.

- Guselkumab [15]: An IL-23 p19 inhibitor, guselkumab proved superior to secukinumab at week 48 for PASI 90, with highly durable responses.

- Risankizumab [14]: Another IL-23 inhibitor, risankizumab achieved significantly higher PASI 90/100 rates than ustekinumab, with convenient q12-week dosing.

- Tildrakizumab [20]: Showed long-term efficacy with favorable safety and drug survival rates.

Safety Profile

IL-23 inhibitors are among the safest biologics in dermatology, with low infection risk and excellent tolerability. IL-17 inhibitors are associated with candidiasis and mild respiratory infections, but serious adverse events remain rare.

Emerging Therapies

Next-generation therapies include oral IL-23 pathway inhibitors (e.g., icotrokinra/JNJ-2113), which aim to combine the efficacy of biologics with the convenience of oral dosing.

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Table 3: Key Biologic Therapies in Psoriasis

Agent	Target	Pivotal Trial(s)	Population / Endpoints	Key Efficacy Results	Safety Profile
Bimekizumab	IL-17A/F	BE SURE, BE RADIANT (NEJM 2021)	Moderate–severe plaque psoriasis	PASI 90/100 rates superior to adalimumab and secukinumab	Oral candidiasis (10–15%), mild/moderate
Guselkumab	IL-23 p19	ECLIPSE (Lancet 2019)	Moderate–severe psoriasis	PASI 90 at week 48 superior to secukinumab	Favorable; low infection risk
Risankizumab	IL-23 p19	UltIMMa-1/2 (Lancet 2018)	Moderate–severe psoriasis	PASI 90: ~75% vs 47% (ustekinumab); durable q12w dosing	Favorable; minimal immunogenicity
Tildrakizumab	IL-23 p19	reSURFACE trials (2017)	Moderate–severe psoriasis	Sustained PASI 75/90 clearance long-term	Good safety; low discontinuation rates
Secukinumab	IL-17A	ERASURE, FIXTURE (2014)	Moderate–severe psoriasis	PASI 75/90 high rates at 12–16 wks; rapid onset	Nasopharyngitis, candidiasis risk
Ixekizumab	IL-17A	UNCOVER trials (2016)	Moderate–severe psoriasis	High PASI 90/100 rates at 12–16 wks; durable	Candidiasis, mild infections

Psoriasis has become a model for precision biologic therapy, with IL-23 and dual IL-17 inhibitors delivering high-level clearance, durable remission, and favorable safety, reshaping long-term disease expectations.

DISCUSSION AND CONCLUSION

The emergence of monoclonal antibodies and biologic therapies has redefined the therapeutic landscape of autoimmune diseases, offering precision immunomodulation where broad immunosuppression once dominated. Across diverse conditions such as pemphigus vulgaris, generalized myasthenia gravis, and psoriasis, biologics have proven transformative by targeting disease-specific pathogenic pathways while sparing global immune function.

In pemphigus vulgaris, B-cell depletion with rituximab has shifted the standard of care from long-term corticosteroid dependency to durable remission, highlighting the central role of autoreactive B cells in disease propagation. The success of rituximab underscores the broader principle that selective elimination of immune effector populations can achieve lasting disease control, a concept echoed in other autoantibody-driven conditions.

In generalized myasthenia gravis, mechanistic insights into complement activation and IgG recycling enabled the development of complement inhibitors (eculizumab, ravulizumab, zilucoplan) and FcRn antagonists (efgartigimod, rozanolixizumab). These therapies provide rapid and sustained symptom relief with tailored dosing regimens, illustrating how biologics can directly modulate disease physiology to deliver meaningful functional improvements in previously refractory patients.

In psoriasis, advances in understanding the IL-23/Th17 axis transformed management from partial clearance with TNF inhibitors to near-complete skin clearance with IL-17 and IL-23 inhibitors. Psoriasis thus serves as a model of how pathway-specific targeting can achieve outcomes once thought unattainable, including long-term disease control with high safety margins.

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Despite these successes, challenges remain. Safety considerations—ranging from infusion reactions and opportunistic infections (PV, gMG) to candidiasis with IL-17 blockade (psoriasis)—necessitate vigilant monitoring. Cost and access are major barriers, particularly in low- and middle-income regions, limiting the global impact of these therapies. Furthermore, the need for biomarkers to predict response and guide patient selection remains unmet across all three diseases.

Looking forward, the trajectory of biologic development points toward next-generation strategies: antigen-specific cell therapies (DSG3-CAART in PV), oral biologic mimetics (e.g., IL-23 inhibitors in psoriasis), and novel complement or FcRn modulators in gMG. These innovations promise to refine efficacy, safety, and convenience, while the eventual integration of precision medicine approaches may enable personalized therapy aligned to individual immune signatures.

Collectively, the experiences from pemphigus vulgaris, generalized myasthenia gravis, and psoriasis illustrate the transformative potential of biologic therapies in autoimmune disease. By shifting paradigms from broad immunosuppression to targeted precision immunotherapy, biologics not only improve patient outcomes but also provide a blueprint for future therapeutic innovation across the autoimmune spectrum.

Future Perspectives

The remarkable success of biologic therapies in pemphigus vulgaris, generalized myasthenia gravis, and psoriasis reflects not only advances in immunology but also the ongoing refinement of biotechnology platforms. As these fields mature, several themes are shaping the next generation of autoimmune therapeutics.

1. Antigen-Specific Cell Therapies

While broad B-cell depletion with rituximab has been highly effective in PV, it is inherently nonspecific. Antigen-targeted approaches such as chimeric autoantibody receptor T cells (CAAR-T) are in early development. DSG3-CAART, for example, aims to selectively eliminate B cells producing pathogenic desmoglein antibodies while sparing protective immunity. Early studies suggest feasibility, though optimization of safety and durability will be critical before clinical adoption.

2. Next-Generation FcRn and Complement Modulators

In gMG, FcRn inhibition and complement blockade have proven transformative. Future therapies may offer oral FcRn inhibitors or subcutaneous long-acting complement blockers, improving convenience and adherence. Additionally, bispecific antibodies targeting both complement and FcRn pathways simultaneously could theoretically provide deeper disease control with fewer injections.

3. Oral Biologic Mimetics

In psoriasis, biologics have set unprecedented efficacy standards, but their parenteral administration remains a barrier. Novel oral peptide agents such as icotrokinra (JNJ-2113), designed to inhibit the IL-23/IL-17 pathway, are demonstrating high levels of skin clearance in early trials, rivaling injectable biologics. If validated, these agents could democratize access and adherence, particularly in resource-limited settings.

4. Bispecific and Multispecific Antibodies

The next wave of monoclonal antibody engineering includes bispecific antibodies capable of targeting two immune pathways simultaneously (e.g., IL-17 + IL-23 dual blockade). Such strategies may provide synergistic efficacy, reduced immunogenicity, and simplified regimens compared with combination therapy.

5. Precision Medicine and Biomarkers

Despite high efficacy rates, not all patients respond equally to biologics. The integration of biomarkers of immune signatures (autoantibody profiles, cytokine networks, genetic susceptibility) may guide treatment

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selection and predict response, enabling a truly personalized approach. In the future, treatment may be individualized not only by disease but also by patient-specific immune phenotypes.

6. Access, Cost, and Sustainability

Perhaps the most pressing future challenge is ensuring equitable access. Biosimilars of rituximab and TNF inhibitors have already improved affordability in some regions. The expansion of biosimilars for IL-17 and IL-23 inhibitors, alongside novel oral small molecules, may help balance innovation with global accessibility.

In summary, future directions in biologic therapy are defined by a dual focus: greater precision (antigen-specific or multispecific targeting) and greater accessibility (oral mimetics, biosimilars). Together, these advances promise to consolidate the biologic revolution while making it more patient-centered, sustainable, and globally impactful.

Conflict of Interest: The authors declare no conflict of interest.

REFERENCES

1. Lee, J. J., & Ballow, M. (2010). Monoclonal antibodies and fusion proteins and their complications: Targeting B cells in autoimmune diseases. Journal of Allergy and Clinical Immunology, 125(4), 814–820.
2. Bachu, B., Padhy, S., Manisha, R. L., Nandabaram, A., Vummarao, T., & Sudhakar, M. (2019). Understanding psoriasis: A comprehensive review of its causes and treatments. Frontier Journal of Pharmaceutical Sciences and Research, 2, 69–72.
3. Joly, P., Maho-Vaillant, M., Prost-Squarcioni, C., et al. (2017). First-line rituximab combined with short-term prednisone versus prednisone alone for pemphigus (Ritux 3): A prospective, multicentre, parallel-group, open-label randomised trial. The Lancet, 389(10083), 2031–2040.
4. Werth, V. P., Joly, P., Mimouni, D., et al. (2021). Rituximab versus mycophenolate mofetil in patients with pemphigus vulgaris (PEMPHiX trial). New England Journal of Medicine, 384(24), 2295–2305.
5. Joly, P., Mouquet, H., Roujeau, J. C., et al. (2024). A single cycle of rituximab for the treatment of severe pemphigus (Ritux 3 long-term outcomes). Journal of the American Academy of Dermatology, 90(2), 340–349.
6. Hebert, V., Murrell, D. F., Werth, V. P., et al. (2021). Rilzabrutinib, an oral Bruton's tyrosine kinase inhibitor, in pemphigus vulgaris (Phase 2). The Lancet, 397(10289), 255–265.
7. PRNewswire. (2022). Sanofi and Principia announce results of PEGASUS phase 3 study of rilzabrutinib in pemphigus vulgaris [Press release].
8. Howard, J. F. Jr., Utsugisawa, K., Benatar, M., et al. (2017). Safety and efficacy of eculizumab in anti-acetylcholine receptor antibody-positive refractory generalized myasthenia gravis (REGAIN): A phase 3, randomised, double-blind, placebo-controlled, multicentre study. The Lancet Neurology, 16(12), 976–986.
9. Muppidi, S., Utsugisawa, K., Benatar, M., et al. (2019). Long-term safety and efficacy of eculizumab in generalized myasthenia gravis. Neurology, 92(21), e2261–e2273.
10. Kulkarni, R., Howard, J. F., Nowak, R. J., et al. (2022). Ravulizumab in patients with anti-acetylcholine receptor antibody-positive generalized myasthenia gravis (CHAMPION-MG): A randomised, double-blind, placebo-controlled, phase 3 trial. The Lancet Neurology, 21(6), 526–536.
11. Howard, J. F. Jr., Bril, V., Vu, T., et al. (2021). Efgartigimod in generalized myasthenia gravis (ADAPT trial). The Lancet Neurology, 20(7), 526–536.
12. Mantegazza, R., Wolfe, G. I., Muppidi, S., et al. (2023). Rozanolixizumab in generalized myasthenia gravis (MycarinG): A phase 3 randomised, double-blind, placebo-controlled trial. The Lancet Neurology, 22(5), 421–432.
13. Patwa, H. S., Bril, V., Muppidi, S., et al. (2023). Zilucoplan in patients with generalized myasthenia gravis (RAISE): A phase 3 randomised, double-blind, placebo-controlled trial. The Lancet Neurology, 22(5), 433–443.
14. Gordon, K. B., Blauvelt, A., Papp, K. A., et al. (2018). Phase 3 trials of risankizumab in moderate-to-severe plaque psoriasis (UltIMMa-1 and UltIMMa-2). The Lancet, 392(10148), 650–661.

ISSN No. 2321-2705 | DOI: 10.51244/IJRSI |Volume XII Issue VIII August 2025

15. Reich, K., Armstrong, A. W., Foley, P., et al. (2019). Efficacy and safety of guselkumab, an IL-23 inhibitor, compared with secukinumab, an IL-17 inhibitor, in moderate-to-severe psoriasis (ECLIPSE): Results from a phase 3, randomised controlled trial. The Lancet, 394(10201), 831–839.
16. Mease, P. J., Rahman, P., Gottlieb, A. B., et al. (2020). Guselkumab in patients with active psoriatic arthritis: A randomized, double-blind, placebo-controlled, phase 3 trial. The Lancet, 395(10230), 1126–1136.
17. Warren, R. B., Blauvelt, A., Bagel, J., et al. (2021). Bimekizumab versus adalimumab in plaque psoriasis (BE SURE trial). New England Journal of Medicine, 385(2), 130–141.
18. Reich, K., Papp, K. A., Blauvelt, A., et al. (2021). Bimekizumab versus secukinumab in plaque psoriasis (BE RADIANT trial). New England Journal of Medicine, 385(2), 142–152.
19. Reich, K., Warren, R. B., Lebwohl, M., et al. (2022). Long-term efficacy and safety of bimekizumab in patients with plaque psoriasis: Results from BE BRIGHT extension study. British Journal of Dermatology, 187(5), 663–672.
20. Reich, K., Papp, K. A., Blauvelt, A., Tyring, S., Vanaclocha, F., Kingo, K., Ziv, M., et al. (2017). Tildrakizumab versus placebo or etanercept for chronic plaque psoriasis (reSURFACE 1 and reSURFACE 2): Results from two randomised controlled, phase 3 trials. The Lancet, 390(10091), 276–288.
21. Menter, A., Strober, B. E., Kaplan, D. H., et al. (2020). Joint AAD–NPF guidelines of care for the management of psoriasis with biologics. Journal of the American Academy of Dermatology, 82(6), 1445–1486.
22. Armstrong, A. W., & Read, C. (2020). Pathophysiology, clinical presentation, and treatment of psoriasis: A review. JAMA, 323(19), 1945–1960.
23. Bugaut, H., & Aractingi, S. (2021). Major role of the IL-17/23 axis in psoriasis supports the development of new targeted therapies. Frontiers in Immunology, 12, 621956.
24. Yang, K., Oak, A. S. W., & Elewski, B. E. (2021). Use of IL-23 inhibitors for the treatment of plaque psoriasis and psoriatic arthritis: A comprehensive review. American Journal of Clinical Dermatology, 22(2), 173–192.
25. Camela, E., Potestio, L., Ruggiero, A., Ocampo-Garza, S. S., Fabbrocini, G., & Megna, M. (2022). Towards personalized medicine in psoriasis: Current progress. Psoriasis (Auckland), 12, 231–250.
26. Sarabia, S., Ranjith, B., Koppikar, S., & Wijeratne, D. T. (2022). Efficacy and safety of JAK inhibitors in the treatment of psoriasis and psoriatic arthritis: A systematic review and meta-analysis. BMC Rheumatology, 6, 57.
27. Schön, M. P. (2019). Adaptive and innate immunity in psoriasis and other inflammatory disorders. Frontiers in Immunology, 10, 1764.
28. Chen, L., Deshpande, M., Grisotto, M., et al. (2020). Skin expression of IL-23 drives psoriasis and psoriatic arthritis development in mice. Scientific Reports, 10, 10269.
29. World Health Organization. (2016). Global report on psoriasis. Geneva: WHO.
30. Bachu, B., Rajaputana, L. M., Palle, A., Sushma, R., Nuguri, S. K., Teja, P., Padala, S., & Padhy, S. (2024). Formulation and evaluation of ointment from hydro-alcoholic leaf extract of Cassia auriculata for psoriasis. International Journal of Research in Pharmacology & Pharmacotherapeutics, 13(3), 342–349. https://doi.org/10.61096/ijrpp.v13.iss3.2024.342-349